

# Aflatoxins – The Hidden Danger of Drought

By: Dr. Kerstin Reiners PhD-Product Manager  
(Mastersecure) EW Nutrition GmbH

ew | nutrition 

Pictures of stunted corn cobs in the corn belt of the Midwest of the United States or the barren wheat fields of Ukraine call world food affairs into mind.

What many of us do not take into consideration is that the current drought does not only affect crop yields – USA's corn yields are supposed to be 13 % lower than in the previous year - , but also heavily affects the quality of corn.

Aflatoxins have received greater attention than any other mycotoxins. Oilseeds, spices, groundnuts, tree nuts, and especially corn are some of the feedstuffs in which this group of mycotoxins most frequently occurs [1]. Produced by fungi of the *Aspergillus* spp. – primarily *Aspergillus flavus* and *Aspergillus parasiticus* - aflatoxins are a group of approximately 20 fungal metabolites. Aflatoxin B<sub>1</sub> - the most toxic of the aflatoxins - is the most potent naturally occurring chemical liver carcinogen known [2]. Due to their demonstrated carcinogenic effects in susceptible animals and their acute toxic effects in humans, the U.S. Food and Drug Administration (FDA) has enforced regulatory limits on the concentration of Aflatoxins in foods and feeds [3].

## The Role of Drought

Weather conditions are an important contributor to mold infection. Although generally designated as a storage mycotoxin, the pre-harvest contamination of feeds with Aflatoxins is very probable if conditions are favorable. Along grains, corn is the most susceptible to Aflatoxin contamination in the United States [4]. The fungus responsible for Aflatoxin is quite common, but its population increases during hot dry weather and nighttime temperatures greater than 70 degrees F. during corn silking stage. Generally speaking, whether the fungi produce aflatoxins depends on drought stress and rainfall, suitability of crop genotype for its climate, insect damage, and agricultural practices [5].

**The great significance for dairy cows - When aflatoxin B, becomes aflatoxin M<sub>1</sub>.** After being ingested by dairy cows through contaminated feed, aflatoxin B<sub>1</sub> is converted into aflatoxin M<sub>1</sub> by enzymatic activities of enzymes primarily found in the liver [6]. The newly formed metabolite will then be excreted in the urine and in the milk of the cow. At this stage, this mycotoxin is no longer only an animal health concern but has become a public health issue! Due to the transmission of aflatoxin M<sub>1</sub> into milk, milk in the European Union, for example, is even discarded with an aflatoxin M<sub>1</sub> level higher than 0,05 ppb [7].

**Aflatoxin B<sub>1</sub>: Silently damaging the cow's liver.** After ingestion of aflatoxin, its toxicity may be exerted in several ways from altered intestinal integrity to immunosuppression, hemorrhage, growth inhibition, and acute liver damage, depending mainly on the amounts at which it is consumed. Less visible to the naked eye is the bio-transformation of aflatoxin in the liver. Amongst other possibilities, this

molecule may bind to liver proteins potentially causing acute aflatoxicoses or it may bind to DNA, a step precursor for aflatoxin-induced liver cancer [5].

None of the abovementioned options positively affect dairy cow's health and performance especially in the period around parturition as cows undergo tremendous physiological changes during this production stage. Liver function is acknowledged to be altered due to metabolic demands especially during late gestation and early lactation. After calving, the initiation of milk synthesis and the rapidly increasing milk production greatly increase demands for glucose synthesis at a time when feed intake has not reached its maximum [8]. Due to this fact many metabolic changes occur which might lead to fat deposition in the liver and contribute to liver failure [9] straining cows extremely sensitive to toxin challenges.

## The Way Around It


Whether you look at it from a milk quality and consumer health perspective or from the perspective of your own herd's health and performance, aflatoxin contamination of the feed must be properly counteracted.

Especially in years with extended risks of aflatoxin contamination of the feed, treatment of already contaminated corn as well as the prevention of continuing mold growth during storage play an important role for producers.

Mold inhibitors, such as organic acids, can help to prevent mold growth during storage when moisture content rises above 12 – 14 % or poor storage conditions are a problem.

**The mold inhibitors prevent the growth of *Aspergillus* spp. during storage, but they are not able to destroy or modify aflatoxins which have already been produced by *Aspergillus* ssp. prior to harvest.**

## How to treat corn which is contaminated with aflatoxins already prior to harvest?

There are several products on the market, mainly containing clay minerals, which have proven effects in binding aflatoxins and preventing their adsorption. But the binding of aflatoxins alone is not enough to face the problems aflatoxin contaminated corn causes! Even if aflatoxins are bound, the liver is still challenged, especially in high performing animals. In order to provide natural protection of the liver and improve hepatic metabolism, there are products on the market which do not only care about the adsorption of aflatoxins but go further. Products which additionally contain selected herbal extracts, essential oils and zinc adsorb aflatoxins on the one hand, but furthermore support the whole metabolism of the cow to reach and retain high performances. Several studies could proof the additional effects of products with a complete formulation of synergistic components. It could be shown that beside binding aflatoxins and other relevant mycotoxins, the products were effective in controlling reproductive problems, immunosuppression, hepatotoxicity and gastrointestinal disturbances related to mycotoxicosis. 

## Literature

[1] Strosnider, H., Azziz-Baumgartner, E., Banziger, M., Bhat, R.V., Breiman, R., Brune, M., DeCock, K., Dilley, A., Groopman, J., Hell, K., Henry, S.H., Jeffers, D., Jolly, C., Jolly, P., Kibata, G.N., Lewis, L., Liu, X., Luber, G., McCoy, L., Mensah, P., Miraglia, M., Misore, A., Njapau, H., Ong, C., Onsongo, M.T.K., Page, S.W., Park, D., Pate, I.M., Phillips, T., Pineiro, M., Pronczuk, J., Schurz Rogers, H., Rubin C., Sabino M., Schaafsma A., Shephard G., Stroka J., Wild C., Williams J.T., Wilson D. (2006). "Workgroup Report: Public Health Strategies for Reducing Aflatoxin Exposure in Developing Countries." *Environmental Health Perspectives*, 114:1989-1903.

[2] Wu, F., Narrod, C., Tiongco, M., Liu, Y. (2011). The health economics of aflatoxin: global burden of disease. IFPRI, Working Paper 4. Accessed March 2012.

[3] Food and Drug Administration (2000). Guidance for Industry: Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed.

[4] Wood, G.E. (1992): Mycotoxins in foods and feeds in the United States. *Journal of Animal Science* 70: 3941-3949.

[5] Wu, F., Khlangwiset P. (2010). Health economic impacts and cost-effectiveness of aflatoxin reduction strategies in Africa: Case studies in biocontrol and postharvest interventions. *Food Additives & Contaminants* 27: 496-509.

[6] Pennington, J. Aflatoxin M<sub>1</sub> in milk. University of Arkansas, Agriculture and Natural Resources. FSA 4018. Accessed March 2012: [http://www.uaex.edu/Other\\_Areas/publications/PDF/FSA-4018.pdf](http://www.uaex.edu/Other_Areas/publications/PDF/FSA-4018.pdf)

[7] Commission Regulation (EC) No 1881/2006 of 19 December 2006 setting maximum levels for certain contaminants in foodstuffs. *Official Journal of the European Union* L 364.

[8] Drackley J.K. Physiological adaptations in transition dairy cows. Department of animal sciences University of Illinois. Accessed March 2012: [http://www.cvm.umn.edu/dairy/prod/groups/cvm/@pub/@cvm/documents/asset/cvm\\_33551.pdf](http://www.cvm.umn.edu/dairy/prod/groups/cvm/@pub/@cvm/documents/asset/cvm_33551.pdf)

[9] Rehage, J. (1996). Investigations about the risk of liver failure in dairy cows with fatty liver in the model of cows with left abomasal displacement. *Habil-Thesis, Hannover School of Veterinary Medicine, Hannover, Germany.*

EW Nutrition USA, Inc.

3227 99th Street

Des Moines, Iowa 50322

Phone: +1 515-251-7430

Fax: +1 515-251-7427

Email: [us.info@ew-nutrition.com](mailto:us.info@ew-nutrition.com)

[www.ew-nutritionusa.com](http://www.ew-nutritionusa.com)